FUNDUS CHANGES
IN
PREGNANCY INDUCED HYPERTENSION

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CERTIFICATE

This is to certify that this dissertation entitled **"*FUNDUS CHANGES IN PREGNANCY INDUCED HYPERTENSION*"** has been done by ***DR.A. ANU SUJATHA*** under my guidance in Department of OPHTHALMOLOGY, Madurai Medical College, Madurai.

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I, **Dr. A. ANU SUJATHA** solemnly declare that the dissertation titled ***"FUNDUS CHANGES IN PREGNANCY INDUCED HYPERTENSION"*** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S.,(Ophthalmology) Branch - III degree Examination to be held in MARCH 2008.

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CONTENTS

INTRODUCTION

Pregnancy induced hypertension, is the commonest form of life threatening complications of pregnancy. Pregnancy is described as the only physiological state in which most physiological parameters are abnormal. The anatomical, physiological and biochemical adaptations that take place in a woman during the short span of human pregnancy are profound. Compared with normotensive gravidas, patients with elevated blood pressure have significantly greater maternal and fetal mortality and morbidity. The clinical and laboratory characteristics of hypertension associated with pregnancy are difficult to differentiate from those of hypertension independent of pregnancy.

DEFINITION

Pregnancy induced hypertension, PIH, is a syndrome of hypertension with proteinuria or oedema or both, occurring after the 20^{th} week of pregnancy, terminating shortly after delivery. The term

‘toxaemia of pregnancy’ was used for over a century to describe PIH. It is no longer used because it erroneously implies that a toxin circulating in the blood is the cause of the disorder. ‘preeeclampsia-eclampsia’ is another term for, PIH, but since the most commonly used term at present is ‘Pregnancy induced hypertension’, PIH, the same shall be used in the subsequent discussions.

According to the ACOG (American College of Obstetricians and Gynecologists) the diagnosis of hypertension in pregnancy is made by any one of the following criteria:

1. A rise of 30 mm Hg or more in systolic blood pressure.
2. A rise of 15 mm Hg or more in diastolic blood pressure.
3. A systolic blood pressure of 140 mm Hg or more.
4. A diastolic blood pressure of 90 mm Hg of more.

These alterations in blood pressure should be observed on at least two different occasions at least 6 hours apart.

Hypertension in pregnancy is classified into the following groups:

1. Pregnancy-induced hypertension
    a. Preeclampsia
    b. Eclampsia

2. Chronic hypertension of whatever cause, but independent of pregnancy.
3. Preeclampsia or eclampsia superimposed on chronic hypertension.
4. Transient hypertension.
5. Unclassified hypertensive disorders.

Each of these forms of hypertension are defined by ACOG as follows:

Preeclampsia: Hypertension associated with proteinuria, greater than 0.3 g/L in a 24-hour urine collection or greater than 1+ in a random sample; generalized edema, greater than 1^{+} pitting edema after 12 hours of rest in bed or a weight gain 5 lb or more in 1 week; or both after 20 weeks of gestation.

Eclampsia: Convulsions occurring in a patient with preeclampsia.

Chronic hypertension: the presence of sustained blood pressures of 140/90 mm Hg or higher before pregnancy or before 20 weeks.

Preeclampsia or eclampsia superimposed on chronic hypertension: The occurrence of preeclampsia or eclampsia in women with chronic hypertension. To make this diagnosis it is necessary to document a rise of 30 mm Hg or more in diastolic blood pressure, associated with proteinuria, generalized edema, or both.

Transient hypertension: The development of hypertension during pregnancy or the early puerperium in a previously normotensive woman whose pressure normalizes within 10 days postpartum. There must be no evidence of preeclampsia.

Unclassified hypertensive disorders: Those in whom there is not enough information for classification.

CLINICAL FEATURES

INCIDENCE

PIH is said to occur in 5 to 15% of pregnancies and about 17.9% of patients with PIH develop convulsions.

DIAGNOSIS OF PIH

To diagnose PIH, a patient should have at least two of the following three clinical findings:

1. **Hypertension:** This is the most significant primary sign. It refers: systolic BP above 140 mm Hg or 30 mm Hg above baseline value; diastolic BP above 90 mm Hg or 15 mm Hg above baseline value. The reading should be made in the same posture at least twice at 6 hours apart (established by American College of Obstetricians and gynecologists)

2. **Generalised oedema:** Initially fluid retension causes oedema in the lower legs. This may progress to massive oedema, anasarca, or pulmonary oedema in severe PIH. Thus excessive weight gain results, the average weight gain in normal pregnancy being 10-12 kg.
3. **Proteinuria:** Protein above 300mg/l in a 24 hrs collection or above 1+ on random samples. This is usually the last of the 3 major signs to appear.

Risk Factors

- Extremes of maternal age (<18 years or > 35 years)
- Primigravida
- Multiple gestations
- Molar pregnancy
- Pre-existing hypertension
- Diabetes mellitus
- Renal disease
- Pre-existing connective tissue disease(systemic lupus erythematosus)
- Vascular disease
- Obesity
- Thrombophilia

- Prior history of preeclampsia or eclampsia
- Family history of preeclampsia or eclampsia
- Hydrops fetalis
- Triploidy
- Sickle cell disease.

Clinical features of mild pre-eclampsia

- Systolic blood pressure(SBP) 140-159 mm Hg and diastolic blood pressure(DBP) 90-109 mm Hg.
- Pedal edema: while mild lower extremity edema is common in 30% of normal pregnancy, rapidly increasing or non-dependent edema may be a signal of developing pre-eclampsia. However, this theory remains controversial and recently has been removed from most criteria for the diagnosis of pre-eclampsia.
- Rapid weight gain as a result of edema due to capillary leak as well as renal sodium and fluid retention.

Clinical features of severe pre-eclampsia, atleast one or more of the following:

- SBP 160 mm Hg and above or DBP 110 mm Hg and above
- Headache of new onset, may be described as frontal, throbbing, or similar to a migraine headache. However, no classic headache of pre-eclampsia exists
- Visual disturbances, typically scintillations and scotomata. These disturbances are presumed to be due to cerebral vasospasm
- Confusion

- Abdominal pain, more commonly epigastric pain is due to hepatic swelling and inflammation, with stretch of th liver capsule. Pain may be constant, and it may be moderate-to-severe in intensity
- Proteinuria more than or equal to 5 g in 24 hours
- Oliguria, ≤400 ml in 24 hours
- Pulmonary edema
- Microangiopathic hemolytic anemia
- thrombocytopenia
- fetal growth retardation
- Oligoamnios.

Clinical features of eclampsia

- Seizures
- Severe agitation
- Unconsciousness for a variable period of time
- Involuntary movements(tonic-clonic seizures) occur
- The relaxation phase of deep-tendon reflexes may be prolonged
- Breathing(respiration) may cease for brief periods(apnea)
- Infrequently, an eye examination may note retinal changes caused by hypertension.

CLASSIFICATION OF PIH

A. Mild

1. BP ≥ 30 / 15 mm Hg rise; ≥ 140/90 mm Hg
2. Proteinuria ≥ 300 mg/24 hr
3. Generalised oedema

B. Severe(imminent eclampsia)

1. BP ≥ 160 / 110 mm Hg
2. Proteinuria ≥ 5g / 24 hr
3. Urine output < 400 ml/24hr
4. Cerebral or visual disturbances
5. Pulmonary oedema or cyanosis
6. Epigastric or right upper quadrant pain
7. impaired liver function
8. thrombocytopenia

As a result of hypertension, the blood vessels throughout the body are affected. But the eye(retina), is the only place where these vascular changes can be directly visualized and assessed. This places the ophthalmologist in both an advantageous and responsible position.

Needless to say, repeated fundus examination of patients with PIH becomes highly mandatory.

AIM OF THE STUDY

To examine the various fundus changes occurring in patients with pregnancy induced hypertension.

To determine if fundus examinations might prove of value in determining whether or not the pregnancy should be terminated.

PATHOPHYSIOLOGY OF PIH

Pathophysiology of pre-eclampsia

Although the exact pathophysiologic mechanism is not clearly understood, preeclampsia can be thought of as a disorder of endothelial function with vasospasm and arterial constriction and relatively reduced intra-vascular volume compared to normal pregnancy.

Usually, the vasculature of pregnant women demonstrates decreased responsiveness to vasoactive peptides such as angiotensin II and epinephrine. Women who develop pre-eclampsia show a hyperersponsiveness to these hormones; their blood pressures (BPs) are labile, and their normal circadian BP rhythms may be blunted or reversed. Pre-eclampsia has been shown to be associated with platelet activation and excessive release of vasoconstricting thromboxance preceding the onset of the disease.

In some cases, light microscopy demonstrates evidence of placental insufficiency associated with abnormalities, such as diffuse placental thrombosis, an inflammatory placental decidual vasculopathy, and/or abnormal trophoblastic invasion of the endometrium. This association

suggests that abnormal placental development or placental damage from diffuse microthrombosis may be central to the development of this disorder. The widespread endothelial dysfunction may manifest in a pregnant woman as dysfunction of multiple organ systems, including the central nervous, hepatic, pulmonary, renal and hematological systems. The decrease in perfusion can manifest clinically as low scores on a biophysical profile, oligohydramnios, and as fetal growth restriction in severe cases.

Calcium deficiency has been implicated as a possible cause of gestational hypertension. In two preliminary studies, women who developed gestational hypertension were found to have significantly lower dietary calcium intake than did pregnant women with normal BP.

Magnesium deficiency has also been implicated as a possible cause of gestational hypertension. Dietary intake of magnesium is below recommended levels for many women during pregnancy.

Eclampsia

The cause of eclampsia is not well understood. Eclampsia may follow pre-eclampsia, if that condition cannot be brought under control. It is difficult to predict which pre-eclamptic women may go on to have seizures the hallmark of eclampsia. There is poor correlation between the

degree of hypertension present in preeclampsia and the ultimate occurrence of seizures.

BIOCHEMICAL CHANGES IN PIH

1. **SODIUM and Water Balance:** there is retention of sodium and water over and above that found in normal pregnancy. In a normal pregnancy, water retention begins at the 13th week, and by the 40th week, it is about 5.0 litres. The normalamount of sodium retention is about 850 m.eq.
2. **Blood Chloride:** Decreased, Normal 95-105 mmol/l.
3. **Uric Acid:** Increased in severe PIH due to inability of the kidney to excrete it. The normal value is 0.12-0.36 mmol/l or 2-4 mg%.
4. **Urea:** Usually not increased; may increase when associated with oliguria. Normal 20-40 mg% or 3.5 to 7.4 mmol/l.
5. **Albumin and Total Proteins:**
    - There is a 25% fall in Plasma albumin concentration (Normal 4.3g%).
    - There is a slight increase in plasma globulin (Normal – 2.7 b %).
    - There is a 50% increase in plasma fibrinogen level (Normal – 250mg%).

NORMAL ANATOMY OF RETINAL VASCULATURE

The central retinal artery and central retinal vein enter and leave the eye at the optic disc. From the disc they run at a superficial plane in the nerve fibre layer. The arteries have a higher red colour. The veins are darker, and more convoluted. Both have a central reflex. The retinal artery forms an end artery circulation. The vessels divide dichotomously and remain superficial until the immediate precapillaries are reached.

THE CAPILLARIES FALL INTO 2 MAJOR GROUPS:

(i) Superficial Capillary net: Remains in nerve fibre layer.

(ii) Deep Capillary net: Runs at a steep angle to a deeper level and lies in the boundary plane between inner nuclear layer and outer plexiform layer.

These 2 capillary nets are connected with anastamotic capillaries. This basic 2 layered pattern of the vascular architecture is modified in certain parts of the retina either by addition of other layers or by reduction to a single layer.

Three Layered pattern:

In the posterior thicker part of the retina the deep net is unaltered, remaining flat, whereas, from as superficial net fewer capillaries run, from arteriole to venule entirely in the nerve fibre layer, and many capillary loops come to run at the internal boundary of the inner nuclear layer. Thus a 3-layered pattern appears. This is particularly well developed in the macular region.

Four Layered Pattern:

In and around the disk, where the nerve fibre layer is thick, yet another most superficial capillary net appears. Capillaries from the superficial layer proper turn superficial to the plane of this layer and breakup into an extremely dense network of capillaries.

Single layered capillary net:

Towards the periphery of the retina, the two-layered pattern becomes intermittent, the deep net being absent in places. Still further towards the periphery the deep net disappears entirely and there is only a single layer.

CALIBRE OF RETINAL VESSELS:

The arteries are narrower than veins. The normal AV ratio is 2:3. But this is variable due to variations in the caliber of the normal veins and

subjective errors in assessing the ratio. There are 2 ways of assessing the calibre of the artery.

1. The calibre of the artery may be compared with that of the average vessel of normal calibre.
2. The calibre of the artery may be compared with that of the vein and expressed as a ratio.

AV CROSSINGS:

The crossings of the arteries and veins are seen in the fundus. In 80% of crossings the arteries lie above the veins. It is never an artery over an artery or vein over a vein. The maximum number of crossings is seen in the superotemporal quadrant. At the point of crossing both vessels share a common sheath. The vein is clearly visible under the artery.

HISTOLOGY:

Only the central retinal artery and its first branches are true arteries. The rest are really arterioles.

Central Retinal Artery: Has the following layers in its walls

Tunica Interna: has

1. A single layer of endothelial cells lining the lumen.
2. A sub endothelial layer of circularly arranged elastic tissue.

3. An internal elastic lamina composed of elastic fibrils.

Tunica Media: Composed of multiple layers o smooth muscle interspersed with elastic fibres.

Tunica Adventitia: It is the thickest layer and composed mostly of collagen with interspersed circular and longitudinal elastic fibres.

B. Arterioles

Tunica Interna: Has

1. A single layer of endothelium.
2. Elastic fibres are very sparse or absent.

Tunica Media: It is ill defined and consists of 2-4 smooth muscle layers. The elastic fibres are sparsely distributed in the media.

Tunica Adventitia: Contains loosely arranged collagen fibres.

C. Capillaries

Their walls are composed of three distinct elements: Endothelial cells, intramural pericytes and basement membrane.

FUNDUS CHANGES IN PREGNANCY INDUCED HYPERTENSION

Both the retinal and choroidal circulations can be affected. As a general rule, retinal changes are liable to occur when systolic pressure is above 160 mm Hg and diastolic above 100 mm Hg. The following changes may be seen:

1. Hypertensive Retinopathy
2. Serous Retinal Detachment
3. Retinal arterial occlusions
4. Ischaemic optic neuropathy
5. Ischaemic papillophlebitis
6. Peripheral neovascularisation

Of these changes hypertensive retinopathy is the commonest.

HYPERTENSIVE RETINOPATHY

Increased diastolic pressure is more important since it indicates a permanent stress upon the vessel wall.

The chief modifying factors of the clinical picture are:

1. Resilience of vessels
2. Duration of hypertension

3. Height of hypertension
4. Treatment

The clinical picture seen in PIH is a classic example of simple hypertension occurring in resilient vessels. The clinical course passes through 3 stages.

Stage I : Spastic stage of arterial irritation.

Stage II : Stage of sclerosis

Stage III : Stage of retinopathy, when oedema, hemorrhage and damage to tissues occur.

Complete recovery usually follows the institution of adequate treatment during the first pre organic stage.

The first visible ocular sign of PIH is generalized attenuation of retinal arterioles. This occurs first in the nasal periphery, then spreads to the disc and becomes generalized. It usually persists till pregnancy is terminated.

The generalized attenuation is accentuated by focal constrictions. Fleeting spasms may occur. If this is persistent, cotton wool spots may appear. The importance lies in the discovery of these early signs of vascular spasm. It is difficult to say when the functional tonic spasm passes into organic sclerosis. But eventually if pregnancy is allowed to continue and if the blood pressure is elevated, retinopathy occurs.

Features of retinopathy:

1. **Hemorrhage:** Usually flame shaped, situated in the nerver fibre layer. Sometimes dot hemorrhages, situated in the deeper layers of the retina may be seen.
2. **Cotton wool spots:** They are due to ischaemia of the nerve fibre layer and accumulation of axoplasmic material.
3. **Retinal oedema:** Due to exudation of fluid.
4. **Oedema residues:** are hard exudates, due to retention of lipid containing proteins.
5. **Micro Aneurysm**
6. **Disc oedema:** May result from ischaemia of arteriolar occlusion and leakage from injured vessels.

RETINA VASCULAR CHANGES IN HYPERTENSION:

The changes occurring in the retinal vasculature because of hypertension may be classified as:

A) Changes in arterioles

B) Changes at AV crossings

C) Retinopathy

A) Changes in arterioles:

They are

(1) Generalized narrowing of arterioles

(2) Generalized sclerosis

(3) Focal constriction of arterioles.

Generalised narrowing of arterioles:

This is often the earliest sign of hypertension. There are 2 ways of assessing arterial narrowing.

1. The calibre of the abnormal vessel may be compared with the average vessel calibre of individuals without hypertension.

2. The calibre of the arteriole may be compared with that of a venule and expressed as a ratio.the normal AV ratio is 2:3

The former way of assessment is better than the latter as alterations in venular calibre will alter the AV ratio. Based on the degree of arterial narrowing there are grades:

GRADE I : The calibre of the arteriole is ¾ of the average calibre of normal arteriole or ½ of the calibre of veins.

GRADE II : The calibre of the arteriole is ½ of the average calibre of normal arteriols or 1/3 of the average calibre of veins.

GRADE III : The calibre of the arteriole is 1/3 of the average calibre of normal arteriole or ¼ of calibre of veins.

GRADE IV : The arteriole is thread like or invisible.

Generalised Sclerosis:

This is indicative of the duration of hypertension. There are 4 grades.

GRADE I: There is increase in the light reflex, mild depression of veins at AV crossing, decreased visibility of veins underlying the arterioles.

GRADE II: The arteriole has burnished copper colour. There is definite depression of underlying vein and almost complete invisibility of portions on veins under artery.

GRADE III: The arteriole has a silver wire appearance. There is invisibility of veins under crossing and distal dilation of veins.

GRADE IV: The arterioles are seen as fibrous cords; there is no visible blood column.

Focal construction of arterioles:

This is seen when the diastolic pressure is more than 110 mm Hg. The calibre of the arteriole abruptly decreases and almost as abruptly increases. This is usually fleeting and disappears when blood pressure decreases.

There are 4 grades of focal construction:

GRADE I: The calibre of the narrowed artery is 2/3 of the calibre of the proximal segment.

GRADE II: The calibre of the artery is ½ of the calibre of the proximal segment.

GRADE III: The calibre of the artery is 1/3 of the calibre of the proximal segment.

GRADE IV: The artery is invisible beyond beyond the point of constriction or visible as fibrous cord.

B) CHANGES AT A-V CROSSINGS:

These are:

1. **Simple Deflection of Vein:** The deflection is usually lateral taking the shortest direction and resulting in an "s" or "z" pattern between crossing vessels – SALU'S SIGN.
2. **Moderate Compression:** The vein appears partially cut; there is tapering of veins at one or both side (GUNN'S SIGN)

   There may be banking of blood in the vein distal to crossing (BONNET'S SIGN).
3. **MARKED COMPRESSION:** the vein appears completely cut, leaving a clear space on either side of the atery.

C) RETONPATHY: (already described)

CLASSIFICATION OF HYPERTENSIVE RETINOPATHY

There are different classification of hypertensive retinopathy.

A) Keith-Wagner-Barker's Classification

GRADE I: Mild narrowing or sclerosis of retinal vessels.

GRADE II: Marked narrowing of vessels with irregularity of the arteriolar lumen and changes at A-V crossings.

GRADE III: Presence of the retinal oedema, cotton wool spots and flame shaped hermorrhages.

GRADE IV: grade 3 with disc oedema.

B) SCHEIE'S CLASSIFICATION:

Here hypertensive and arteriosclerotic changes are separately classified.

Classification of Hypertensive changes

STAGE I: Slight arteriolar narrowing.

STAGE II: Marked narrowing with focal constrictions

STAGE III: Stage 2 with haemorrhages, cotton wool spots and exudates.

STAGE 4: stage 3 disc oedema

Classification of arteriosclerotic changes

STAGE I: Increased light reflex and AV nicking.

STAGE II: More pronounce than stage 1

STAGE III: Copper wire appearance

STAGE IV: Silver wire appearance

SEROUS RETINAL DETACHMENT

Serous retinal detachment is seen is about 1% of patients with severe PIH and in about 10% of patients with eclamptic convulsions.

PATHOPHYIOLOGY:

Serous retinal detachment results from choroidal ischaemia. The choroidal vascular tone is controlled primarily by the sympathetic nervous system. The choroidal arterioles undergo constriction in response to systemic hypertension; however increased blood pressure can overcome the compensatory tone of sympathetic response and vascular damage can result. The causes of choroidal occlusion and hypertensive choroidopathy are:

1. Ocular sympathetic derangement
2. Fibrin platelet occusion of the chroidal arteries and choriopillaries which may occur as a part of DIC (disseminated intravascular coagulation)
3. Embolic occlution originating from the products of conception on an immunologic basis.

The multifocal areas of choroidal vascular occlusion produce ischaemic changes in the overlying retinal pigment epithelium and outer retinal layers, leading to exudative retinal detachment.

FEATURES

Exudative RD is usually bilateral and often bullous. It is always associated with a generalized oedema frequently marked on the face and

lids. Retinal striae may be the first sign, followed by focal accumulation of subretinal or sub pigment epithelial exudate in the posterior pole. The predominant location of choroidal vascular insufficiency in macula is caused by the increased number if large diameter ciliary vessels and dense chorio capillaries at this site. The detachment may progress to involve the entire retina and is usually associated with yellow-white deposits at level of RPE.

FLUORESCEIN ANGIOGRAPHY

Fluorescein angiography is used to study the status of the Retinal vasculartur e.

In the early phase, there is choroidal nonfilling

In the mid phase, persistent choroidal nonperfusion and hyperfluorescence.

In the late phase, extravasation of dye into the subpigment epithelial and subretinal spaces.

Although teratogenic effects have not been identified, Flurescein angiography should be avoided in pregnant women unless absolutely necessary. Also of note, the flurecein will be transmitted to Breast milk in lactating women.

Indocyanine green has also been used in various studies instead of fluorescein.

PROGNOSIS:

Though retinal detachment can cause marked loss of visual acuity, most patients experience full resolution of the detachment spontaneously with return to normal visual function within the first few weeks postpartum. Retinal detachment is not an additional risk factor for adverse fetal outcome.

SEQUELAE:

Some patients with retinal detachment have residual macular retinal epithelial change. Years later, these changes can mimic a macular dystrophy or tapeto retinal degeneration.

RETINOPATHY AND RETINAL DETACHMENT:

They can occur independent of one another. In most of the reports of retinal detachment in literature, there was no evidence of angiospasm at all.

RETINAL ARTERIAL OCCLUSIONS:

Purtscher's like retinopathy, a picture of multiple retinal arteriolar occlusions, can occur within 24 hours after childbirth. The fundus shows white retinal patches characteristic of ischaemia, and occlusive intraretinal hemorrhages. On resolution the acute process, there is resultant arteriolar narrowing and optic disc pallor. Visual defects occur compatible with the areas of occlusion.

Cause: The arterial occlusions are thought to be caused by complement induced leukoemboli. These emboli may be related to abnormal levels of clotting and clot inhibiting factors. The abnormalities seen are:

1. Increase of plasma factor VIII
2. Deficiency of protein S, a vitamin K dependent plasma protein which inhibits the clotting cascade

ISCHAEMIC OPTIC NEUROPATHY

Visual loss can occur as a result of widespread capillary occlusion in which the optic nerve is also involved. Selective optic nerve involvement is rare. Anterior ischaemic optic neuropathy can occur due to impairment of blood supply to the prelaminar portion of the optic nerve head.

MANAGEMENT OF PREGNANCY INDUCED HYPERTENSIVE RETINOPATHY

This depends on the stage of retinopathy.

STAGE OF ANGIOSPASM AND SCLEROSIS

During this stage the pregnancy can be safely allowed to continue. The patient is advised adequate rest, sedation, good protein diet and salt restricted diet. The fluid balance should be well maintained and antihypertensives used to control hypertension.

The angiospasm disappears within a few days or weeks of delivery.

STAGE OF RETINOPATHY

Once retinopathy sets in, with hemorrhages, and exudates, particularly when disc oedema is present, the pregnancy should be terminated. This should be done at the earliest possible time to avoid endangering the life of the mother and the fetus. The retinopathy subsides after termination of pregnancy, with frequently complete restitution of visual acuity. But the organic vascular sclerosis persists.

PROGNOSIS

Prognosis is very much less grave than in an equally severe primary hypertensive retinopathy. If it had once been present and had subsided, the likelihood of recurrences in future pregnancies is not great, provided relief has been obtained before the vascular system has been permanently damaged.

REVIEW OF LITERATURE

HYPERTENSIVE RETINOPATHY:

Only 4 years after the invention of the opthalmoscope by Von Helmholtz, Von Graefe in 1855 first described the retinopathy of pregnancy. Silex in 1895 reported the first large series of 35 cases and he expressed the belief that retinopathy occurred once in about 3,000 pregnancies.

Miller was the first obstetrician to attempt to correlate changes in the fundus with Pregnancy induced Hypertension (PIH). He did his own ophthalmoscopy but had an ophthalmologist corroborate his findings in most cases. In his opinion, retinopathy was not only an indication for immediate termination of the pregnancy but was also an indication for sterilization to prevent future pregnancies.

In a large number of patients with PIH at the Boston Lying – in Hospital, Chency (1924) found narrowing of the retinal arterioles in most of those who had marked hypertension, and the constriction was dependent only on the hypertension and not on whether the condition was acute toxaemia or nephritis. He reasoned that in PIH, the vaso constriction is sudden and retinopathy often develops because the retina doesn't have time to compensate for the diminished blood supply;

whereas in arteriolar sclerosis of long standing the constriction of the arterioles is often more pronounced but the frequency of retinopathy is much less because the change is slow in developing and the retina had time to compensate.

Masters in 1933, reported his findings in the routine ophthalmoscopic examination of 269 patients. He found a generalized uniform constriction of retinal arterioles in all patients whose systolic blood pressure was elevated to 150 mm Hg.

Wagener found spastic constriction of the retinal arterioles in about 70% of women with pregnancy induced hypertension and considered it to be usually the primary sign of retinal involvement. In about 60% of the patients, the spastic lesions disappeared when pregnancy was terminated and the blood pressure returned to normal. In about 40% of his patients organic lesions developed in the arterioles and elevated blood pressure usually persisted. He showed by biopsy and at necropsy that the arterioles throughout the body were permanently damaged in patients with retinopathy and expressed the belief that the majority of them would have persistent hypertension.

Gibson reported 39 cases of PIH; in 8 the fundus was normal; in 20 there was pre organic arteriolar constriction, and in 11 organic changes developed.

Sadowsky correlated increasing vascular changes with increasing severity of PIH and foetal mortality and used progressive retinal arteriolar changes as a guideline for termination of pregnancy.

Schultz and 0' Brien in 46 cases found normal fundus in 9, arteriolar spasm in 13, vascular sclerosis in 12 and retinopathy in 12.

Borras reported fundus findings in 150 cases in 1960. the findings were : attenuated retinal arterioles in 77.4%, hemorrhages and exudates in 8%, and disc oedema in 4%.

Capoor et al in 1995 reported the presence of white centered retinal hemorrhages in patients with PIH.

SEROUS RETINAL DETACHMENT

Von Graefe first reported spontaneous retinal detachment in PIH in 1855. Although retinal detachment is now a well recognized complication of PIH, it is very uncommon.

Fry noted a 1.2% incidence in preeclampsia and 10.4% in eclampsia.

Hallum reported 6 cases in 300 patients with PIH.

Mittelstrass and wolfhagen reported 1 case in 973 cases of PIH.

Kronenberg reported 2 cases in 20,538 pregnancies and bosco reported 1 case in 18,524 pregnancies.

In a study conducted at Los Angeles county university of California during a 5 year, only 2 cases were reported in 62,832 deliveries.

Verderame in 1911, was the first to suggest that retinal detachment was caused by pathological changes in the choroid. Previously it was thought that both the choroids and the retina played a role. The role of choroids in the aetiology of retinal detachment was proved by doing colour fluorescein angiography in patients with retinal detachment by Kenny etal in 1972. in 1996, valluri et all performed diagnostic indocyanine green angiography in patients with PIH and established the role of the choriodal vasculature in the pathogenesis of exudative retinal detachment. They reported nonperfusion in the early phases of the angiogram and staining of the choroidal vasculature with subretinal leakage in the late phases of the angiogram and multiple punctuate areas of blocked fluorescence.

Retinal detachments have generally been observed in the absence of angiospasm though both can coexist.

RETINALPIGMENT EPITHELIAL TEAR

A case of RPE tear following PIH has been described in a 28 year old woman by Menchini et al in 1995. This patient developed a RPE tear in the macular region after abruptio placenta and delivery, which presumably followed a RPE detachment.

MATERIALS AND METHODS

The study was conducted at Govt. Rajaji Hospital Madurai, Maternity section, on 100 patients with pregnancy induced hypertension.

THE PATIENTS WERE GROUPED ACCORDING TO:

1. Age
2. Parity
3. Severity of hypertension
4. Grade of fundus changes

For all the patients bed side visual activity was tested in the maternity ward as all these patients could not be shifted to the ophthalmology department prior to delivery. External ocular examination was done. The pupils were dilated with tropicamide and detailed fundus examination was done in the maternity ward. The blood pressure was recorded.

Routine urine analysis for the presence of protein and sugar was done. Blood biochemical investigations like blood urea, s.creatinine,

s.uric acid and total proteins were done. Routine hematological investigations like hemoglobin and platelet count were done. After delivery, the patients were reviewed in the ophthalmology department. A completer ophthalmic examination was carried out. Visual Acuity was tested with Snellens chart. Central visual fields were examined with tangent screen. After papillary dilatation, detailed fundus examination was done. The findings were documented with fundus photography.

OBSERVATION

Table I

Incidence of PIH according to age

≤ 20 years	21-25 years	26-30 years	31-35 years	> 35 years
14	62	16	4	4

PIH affects younger age groups. This is in accordance with the observation of Williams, Krishna menon, Bhasker Rao and others.

Table II

Grouping of patients according to parity

Primigravida	Multigravida
60	38

PIH is more common in Primigravida than Multigravida. This observation is in accordance with the observation made by various authors both western and Indian – Williams, Chesley, Lewis, Mudaliar and Menon, Dawn.

Table III

Grouping according to severity of hypertension

Group A (mild PIH) Diastolic BP ≤100mmHg	Group B (severe PIH) Diastolic BP >100mmHg
83	17

Table IV

Grouping according to fundus changes

In this series of 100 patients, only hypertensive retinopathy was seen. Keith – Wagner's classifications is taken as a guide to classify the fundus changes

Normal	Grade I	Grade II	Garde III	Grade IV
20	43	32	4	1

Table V

Incidence of fundus changes in mild and severe PIH

	Mild PIH Diastolic BP ≥ 100mm Hg.	Severe PIH Diastolic BP>

		100mm Hg.
Normal	20	0
Grade I	43	0
Grade II	20	13
Grade III	0	3
Grade IV	0	1
Total	83	17

On the whole fundus changes were seen in 80% while 20% had normal fundus, 75% have mild degree of fundus changes. 5% have severe degree of fundus changes.

It is seen here that a greater percentage of fundus changes is observed in patients with higher levels of hypertension. This observation is in accordance with the statement of Duane and Duke Elder, who state that increased diastolic pressure is the most important factor for the development of hypertensive retinopathy since the vacular changes in the retina are caused directly by the elevated pressure.

The same observation was also made by kurt et al who found fundus changes in 41% of patients with a B.P of less than 150/100 and 175/125 mm hg, and in 98% with B.P. above 175/125 mm hg.

ANALYSIS AND DISCUSSION

Patients with normal fundus are omitted in this discussion. This done not mean repeated fundus examinations are not required. As mentioned earlier, repeated fundus examination of PIH patients is a must,

as the patient may develop a fleeing spasm at any time. In this study, repeated fundus examination of patients with normal fundus was possible only in 15 cases, as most of the patients had received no antenatal care, getting admitted only a day or two prior to delivery. Arterial attenuation was noticed in 4 patients with sustained elevation of BP during subsequent examination. The rest had normal fundus till delivery.

These patients were put on bed rest and treated with mild antihypertensive drugs. The blood pressure came down to normal after delivery. The treatment was discontinued after the BP was stabilized at normal.

The cases with fundus changes are discussed in detail with fundus photographic illustrations.

A) Grade I HYPERTENSIVE RETINOPATHY

Relation to parity

Total	Primigravida	Multigravida
43	29	14

Relation to Age

≤ 20 Yrs	21 to 25 Yrs	26 to 30 Yrs	31 to 35 Yrs	More than 35 Yrs
6	29	5	1	2

Relation to severity of hypertension

Total	Group A ≤ 100 mmHg	Group b > 100 mmHg
43	43	0

Out of the series of 100 cases, 43 cases presented with grade I hypertensive retinopathy. Thus grade I changes were the commonest. Both Primigravida and Multigravida are seen in this group.

Grade – I Hypertensive retinopathy patients have diabetes B.P less then 100mm Hg

CASE I

Rubi, a 26 year old women Primigravida 1,(IP No.15918/07) was admitted at term with history of labour pains. Physical examination

showed a BP of 150/100 mm Hg, and bilateral pedal oedema. On abdominal palpation fetal parts were felt. External ocular examination was normal.

Ophthalmoscopic examination revealed a normal disc. There was generalized attenuation of arterioles which were pale and straightened. The arterio venous ratio was 1:3. The macula was normal. There were no haemorrhages or exudates.

Urine analysis showed no proteinuria. The blood chemistry results were Blood urea – 24 mg%, S.creatinine 0.8 mg%, S.uric acid 4.4 mg%.

She was put on antihypertensives. She delivered live female baby. Her postpartum BP was 140/90mmHg. Fundus examination after delivery showed that the arterioles were still mildly attenuated. The arteriovenous ratio was 1:2.

CASE II

Eswari, a 25 year old Gravida2para1 (IP no 41461/07) was brought to the hospital with history of generalized oedema and labour pains. On

examination, her BP was 140/100 mmHg. There was pedal oedema and abdominal wall oedema.

On fundus examination the disc was found to be normal. There was generalized arteriolar narrowing with an AV ratio of 1.3. The foveal reflex was normal. The patient was started on antihypertensive drugs, but the BP remained static at 100 mmHg diastolic. The urine output was adequate. As the patient was full term, the pregnancy was terminated with syntocinion drip. After induction the patient delivered a live male baby weighing 2 kg. The investigation results were: urine albumin – nil, Blood urea 22mg%, S.creatinine 0.8mg%, S.uric acid 5.6 mg%. The postpartum BP was 120/80mmhg and there were no convulsions in the postpartum period.

Fundus examination at the time of discharge revealed normal arteriolar caliber with a normal AV ratio of 2:3

DISCUSSION

In grade I hypertensive retinopathy, if no organic vascular changes have occurred, the arterial attenuation is totally reversible after delivery,

there is no threat to the maternal life, vision or foetal life. Most of the patients in this study delivered spontaneously. Hence, with grade I hypertensive retinopathy, pregnancy can be safely allowed to continue with control of maternal hypertension. In case II, the pregnancy was terminated for purely obstetric reasons.

B) GRADE II HYPERTENSIVE RETINOPATHY

Relation to parity

Total	Primigravida	Multigravida
32	20	12

Relation to Age

≤ 20 Yrs	21 to 25 Yrs	26 to 30 Yrs	31 to 35 Yrs	More than 35 Yrs
5	20	6	1	1

Relation to severity of hypertension

Total	Group A ≤ 100 mm Hg.	Group B > 100 mm Hg.
32	20	12

Out of the 100 cases, 32 cases showed grade II hypertensive changes. Both Primigravida and Multigravida are seen in this group but Primigravida predominate.

Case I

Punithavathy, a 20 year old Primigravid woman (IP No.: 423840), was admitted at term in the maternity ward on physical examination revealed a B.P of 160/110 mm Hg and bilateral petal edema had antepartum eclampsia.

Ophthalmoscopic examination showed generalized attenuation of the arterioles as well as focal constriction. The arterioles were pale, straightened and showed acute angled branching. The disc and macula were normal. There were no haemorrhages or exudates.

Her urine analysis showed no proteinuria. Her other blood investigations were: Blood urea 84 mg%, S.Creatinine 1.3mg.%, S.uric acid 4.4 mg%.

The patient was advised bed rest and treated with antihypertensive drugs. Repeat fundus examination showed the same picture. Pregnancy terminated. The patient delivered a live female baby naturally. The post partum BP was 130/90 mm Hg. She was discharged after 3 days. Fundus examination at discharge showed the same finding as before. Fundus – normal after 4 weeks.

Case II

Sulochana, 25 year old woman, grvida 2, para1, (IP No: 423273) was admitted in the 7th month of pregnancy with the history of one episode of generalized convulsions at home. There was no history of PIH in the previous pregnancy. There was no history of systemic hypertension as well as history of seizure disorder. On examination her BP was

160/110 mm Hg. Abdominal examination showed an uterine enlargement of 28 weeks. Foetal heart sounds were normal. There was no pedal oedema.

Ophthalmoscopic examination showed normal disk. There was severe arterioler narrowing with a AV ratio of 1:4 AV crossing changes (salu's sign) were prominent. The macula was normal. There were no hemorrhages or exudates. Her investigation were urine albumin – Nil, blood urea 22mg%, S.creatinine 0.8 mg%, S.uric acid 5.6mg%. After admission, the patient had another attack of generalized convulsions she was sedated and started on antihypertensive drugs, after the 2nd episode of convulsions there were no further convulsions in the hospital. Her BP was stabilized to 150/100 mm Hg. The patient was discharged and asked to review every 2 weeks.

On discharge, her fundus picture showed arteriolar attenuation with AV crossing changes and AV ratio of 1:3.

This patient was lost to follow-up.

DISCUSSION:

Grade II hypertensive retinopathy is characterized by the development of certain amount of organic vascular sclerosis. Hence the arteriolar attenuation is not totally reversible after delivery. Like grade I patients, these patients also normally have safe delivery. But with persistence of hypertension, hemorrhages and exudates can occur in the retina. So these patients should be observed very closely both obstetrically and opthalmoscopically.

C) GRADE III HYPERTENSIVE RETINOPATHY

Relation to parity

Total	Primigravida	Multigravida
4	1	3

Relation to Age

≤ 20 Yrs	21 to 25 Yrs	26 to 30 Yrs	31 to 35 Yrs	More than 35 Yrs
0	1	2	1	0

Relation to severity of hypertension

Total	Group A ≤ 100 mmHg	Group B > 100 mmHg
4	0	4

Out of the 100 patients, 4 patients presented with Grade III hypertensive retinopathy, of which 1 was Primigravid and 3 Multigravid.

Case I

Margret Mary, a 27 years old Primigravid woman (IP. No: 500280) was brought to the hospital with complaint of generalized convulsions and defective vision. She has 2 doses of T.T injection and antenatal check up.

Diagnosed to have PIH. But she has not taken regular treatment.

On examination her B.P was 190/120. There was abdominal well oedema and pedal oedema.

On ophthalmic examination the anterior segment was normal in both eyes. Both pupil were 3mm in size, reacting briskly to light. Defective vision more in left eye then right eye.

Ophthalmoscopic examination showed disc margins – normal. Generalised arterior narrowing 1:4 with focal constriction. Mild retinal oedema present. Superficial hemorrhages present. FR Dull left eye showed superfishal flame shaped hemorrhages superotemporal to disc. Right eye also having superficial hemorrhages temporal to disc. Suggestive of grade III hypertensive retinopathy.

Blood investigations shows urea 33 mg/dl, Sr.Creatinine 1.oo mg/dl, Uric acid 15.3 mg/dl. Uric acid was above normal.

Uterus was 30 weeks in size. Patient had AP Eclampsia and fundus showed grade III hypertensive retinopathy, so ARM done and labour induced. She delivered a dead born male baby of 1.75 kg weight. Then daily fundus examination done. Macular oedema resolved rapidly. B.P was brought under control vision was 6/18 pNIG, 6/24 NIG.

Then review at ophthal op after a month showed good improvement in vision to 6/9 macular oedema resolved. Review after 45 days showed mild arteriolar narrowing. But hemorrhages resolved.

Patient was advised to have regular antenatal check ups and regular treatment future pregnancy.

Case II

Jeyamani, a 24 year old second gravid women, (IP No: 423770) had severe PIH in the 34th week characterized by a history of seizures, severe headache, bilateral pedal oedema and hypertension. Her BP was 200/140 mmHg. During the previous pregnancy, the patient had an abortion in the 3rd month. Fundus examination showed normal disc, retinal oedema in the posterior pole and generalized & focal constrictions of arterioles in both eyes. In the left eye, there were superficial flame shaped haemorrhages, nasal to the disc. One haemorrhage was pale centred, capoor et al also reported the occurrence of white centred retinal haemorrhages in PIH. The investigations done were: Urine albumin-nil, blood urea 30mg%, S.creatinine 0.9 mg%, and uric acid 3.4mg%.

Termination of pregnancy was advised. The pregnancy was terminated by induction following which the patient delivered a live male baby weighing 2 kg.

The postpartum BP was 140/100 mmHg. At discharge, fundus examination showed adsence of retinal oedema, resolving hemorrhage

and persistence of generalized arteriolar narrowing. There were however no focal angiospasms.

Discussion:

Prompt recognition of hypertensive retinopathy and urgent termination is a must to prevent progression to grade IV hypertensive retinopathy. Termination of pregnancy is followed by a favourable outcome to both mother and foetus.

D) GRADE IV HYPERTENSIVE RETINOPATHY

Relation of parity

Total	Primigravida	Multigravida
1	0	1

Relation of Age

≤ 20 Yrs	21 to 25 Yrs	26 to 30 Yrs	31 to 35 Yrs	More than 35 Yrs

0	0	1	0	0

Relation to severity of hypertension

Total	Group A ≤ 100 mmHg	Group B > 100 mmHg
1	-	1

Out of the 100 patients only 1 patients presented with grade IV hypertensive retinopathy. The patients was Multigravid belonged exclusively to group B hypertension. The incidence in group B was 4.3%.

Case I

Selvarathi, a 30 year old women, gravida 3 para, (IP No: 425094) had severe PIH in the 3rd trimester, characterized by hypertension, proteinuria, generalized oedema and headache. The first pregnancy which was 10 years back was uneventful with no history suggestive of PIH. Second pregnancy 2 yrs back uneventful no PIH. On examination her BP was 180/130 mmHg.

On fundus examination, she had bilateral, hyperaemic, oedematous discs with blurred magins. There was generalised arteriolar narrowing.

The veins were superficial haemorrhages close to the disc. Large cotton wool spots were also seen.

The macula was normal

Her urine analysis showed 1+ proteinuria. The blood urea was 22mg%, S.creatinine 0.8mg% and S.uric acid 4.4mg%. Termination of pregnancy was advised. The pregnancy was terminated by induction following which the patient delivered live female baby.

Fundus examination a week later showed that there was no disc oedema or retinal oedema. The arterioles were still attenuated. There were superficial haemorrhages close to the disc, as well as cotton wool spots. Her BP was 130/90mmHg.

Discussion:

Pregnancy induced hypertension is more common at both extremes of reproductive age, (<20, >30) (Dewhurst, Williams, Mudaliar, Menon) Multiple pregnancy is one of the risk factors for the development of PIH. In this study, both patients with grade IV changes had twin pregnancy. Grade IV retinopathy occurs in the presence of severe uncontrolled hypertension and is more common in women who have received inadequate antenatal care. It this study both patients had a diastolic BP of 130 mmHg and more. But on urine analysis, both patients had only 1+

proteinuria. The amount of albuminuria is said to be so variable that it is of no prognostic value. Both patients also had no convulsions and no decrease in the urine output. The blood investigations were normal in both. Thus it was found that the degree of eye changes more closely followed the severity of hypertension than any other single laboratory or clinical sign.

Grade IV hypertensive retinopathy if allowed to persist, carries with it a very grave prognosis for both mother and foetus. The pregnancy should be terminated at the earliest possible time to avoid endangering the life of both. Though the obstetricians have various criteriae for terminating pregnancy, fundus examination is one of the most valuable examinations. In both patients in this study, there was no other criteria for terminating pregnancy like, uncontrolled convulsions, renal failure, hypertensive encephalopathy, pulmonary oedema etc. the only clue to the seriousness of the disease was the fundus finding of grade IV hypertensive retinopathy. This reemphasises the need for fundus examination of PIH patients.

SUMMARY

1. 100 Patients with PIH attending the Govt. Rajaji hospital, obstetrics department were studied. The incidence of PIH, at Government Rajaji hospital, Madurai is about 5.1% per year.
2. The fundus of these patients was examined and data analysed for age, parity, severity of hypertension and fundus changes.

3. The age group of the patients varied from 17 years to 37 years. The maximum number of patients were in the age group of 21 – 25 years.
4. Pregnancy induced hypertension was more common in Primigravida than Multigravida.
5. No other fundus change other than hypertensive retinopathy was seen.
6. Normal fundus was seen in 20% of patients.
7. 43% showed grade I hypertensive changes.
8. 32% showed grade II hypertensive changes.
9. 4% showed grade III hypertensive changes.
10. 1% showed grade IV hypertensive changes.
11. It was found that the frequency and degree of eye changes more closely followed the severity of hypertension than any other single laboratory or clinical sign.
12. Most of the patients with grade I and grade II hypertensive retinopathy had uncomplicated normal delivery of live babies. There was only one case of foetal death.

13. Among patients with grade III and grade IV hypertensive retinopathy (total 5) pregnancy was terminated in 5 patients. There was no maternal death, but 1 cases of foetal death were seen.
14. In all of the cases wherein pregnancy was terminated fundus examination was greatly contributory.

CONCLUSIONS

1. Daily Ophthalmoscopic examinations during hospital stay are another important aid to the obstetricians in the management of patients with PIH.
2. The development of hypertensive retinopathy is directly related to the severity of hypertension.
3. It is relatively safe to allow patients with grade I and II hypertensive retinopathy to continue the pregnancy with treatment.
4. In patients with grade III or IV hypertensive retinopathy, the pregnancy should be terminated irrespective of the period of gestation.
5. Early interruption of pregnancies result in more living babies even though some will be premature.
6. When Ophthalmoscopic examination is done carefully every day, it has proven to be of unquestionable value in the reduction of maternal mortality and foetal mortality.

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PROFORMA

Name : Age : IP:NO

Parity : Period of gestation :

Diagnosis

HISTORY

H/O PIH in previous pregnancy

H/O systemic hypertension

H/O renal dysfunction

H/O epilepsy

H/O blurring of vision

H/O headache

H/O epigastric pain

PHYSICAL EXAMINATION

Level of consciousness

Pedal oedema +/-

Abdominal wall oedema +/-

Anasarca +/-

Convulsions +/-

Body weight

BP prior to delivery

OCULAR EXAMINATION PRIOR TO DELIVERY

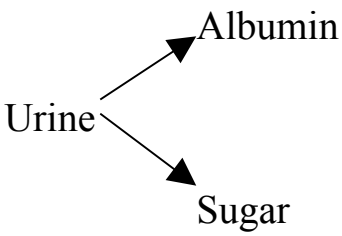
Bedside vision

papillary reaction

Fundus examination

INVESTIGATIONS

Urine output



Blood Hb%

Platelet count

Blood urea

S.Creatinine

S.Uric acid

S. total proteins

S.albumin

S.globulin

NATURE OF DELIVERY

Spontaneous

Induced (terminated)

RESULT AFTER TERMINATION

Maternal mortality

Foetal mortality

B.P. AFTER DELIVERY

OCULAR EXAMINATION AFTER DELIVERY

Anterior segment examination

Visual acuity with snellen’s chart

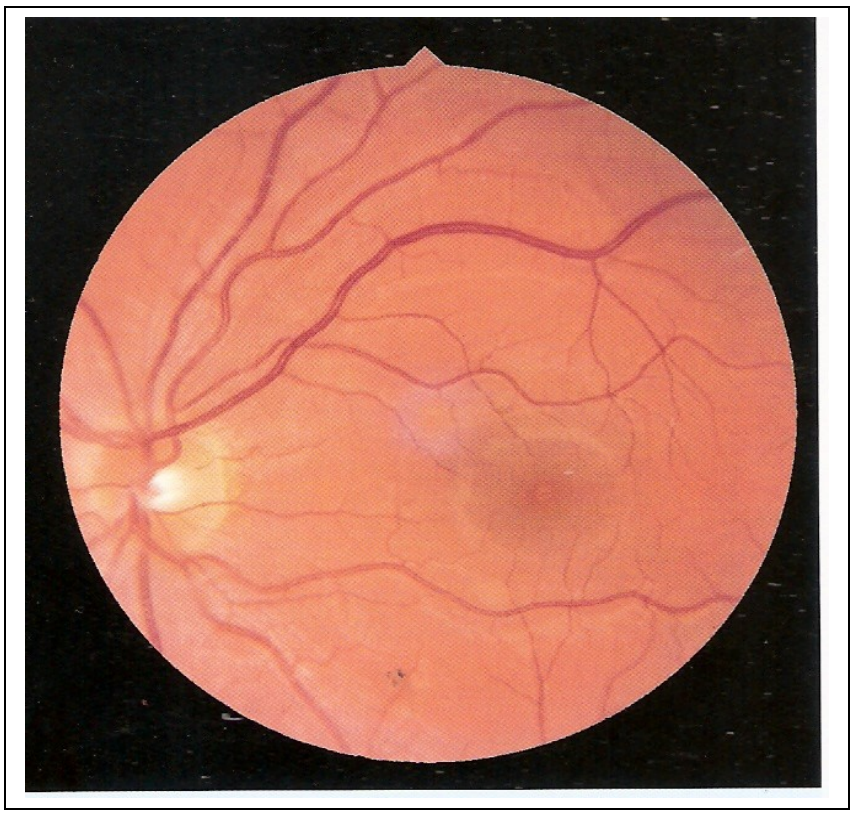
Central fields

Refraction

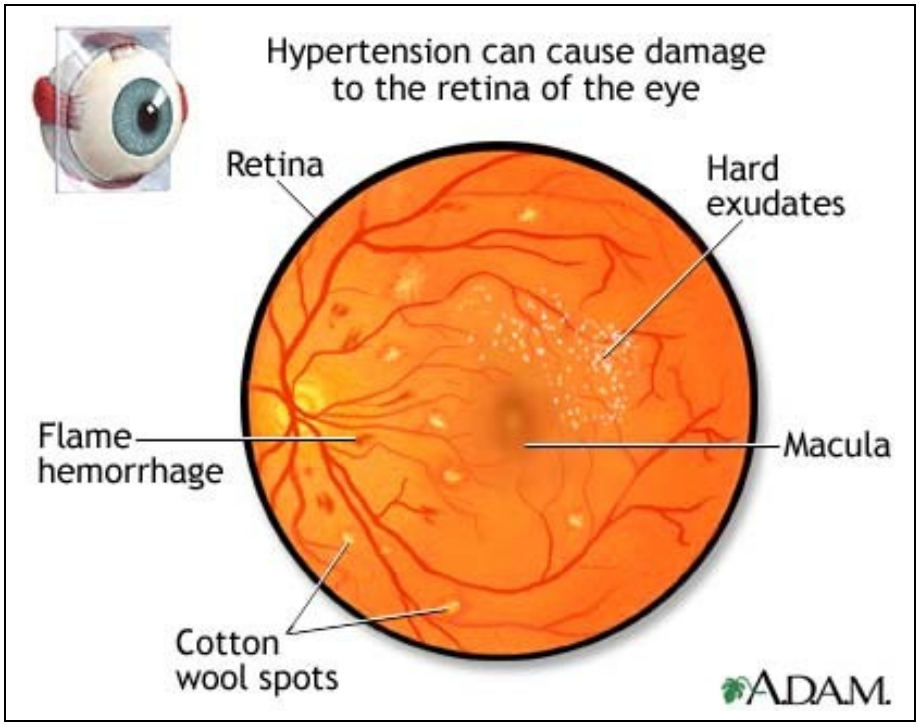
Fundus examination

FUNDUS PHOTOGRAPHY

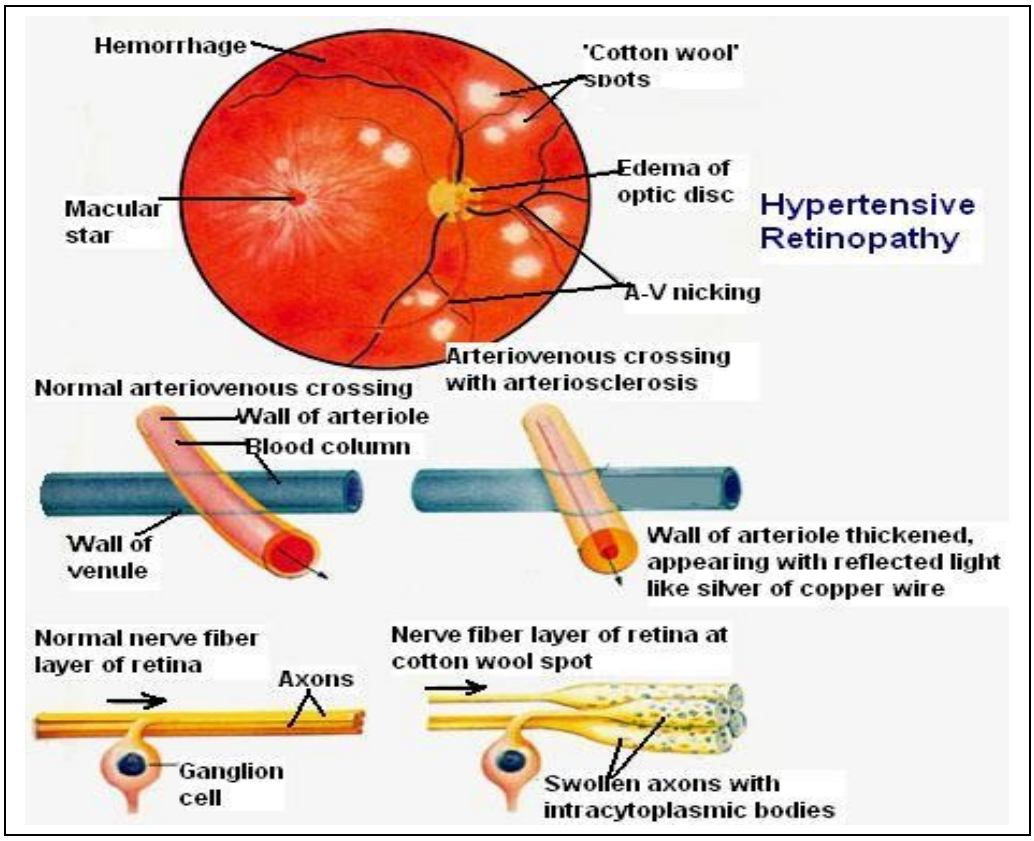
NORMAL FUNDUS

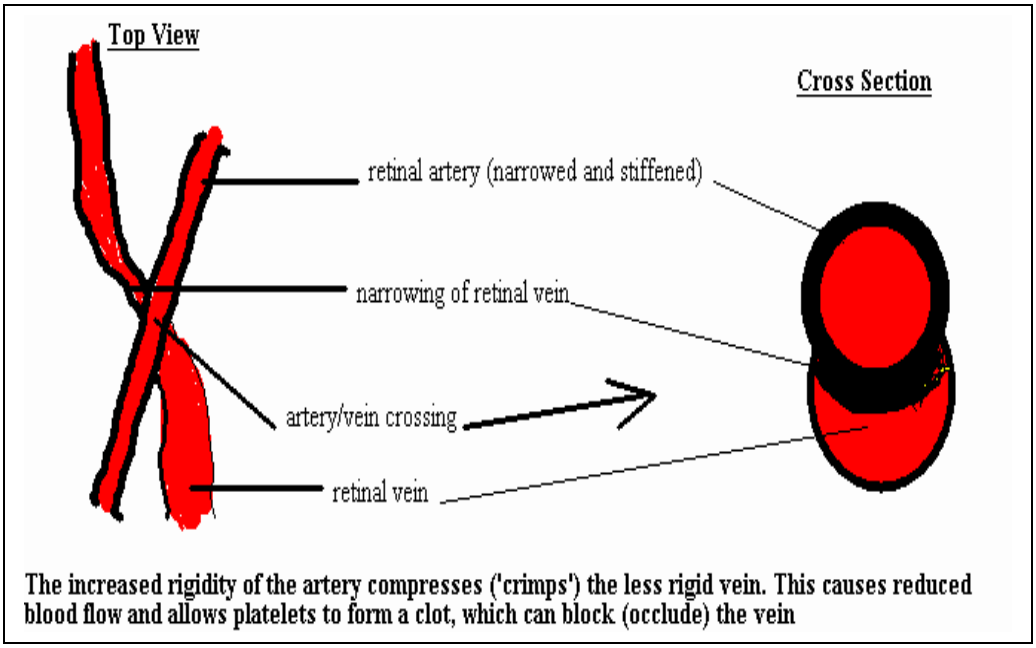


FEATURES OF HYPERTENSIVE RETINOPATHY

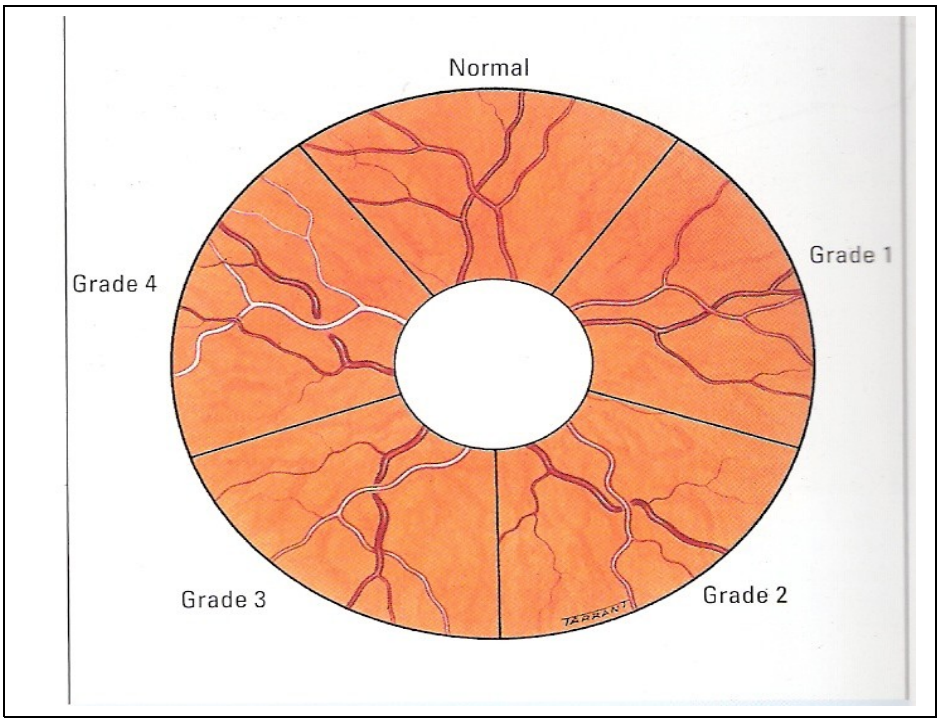


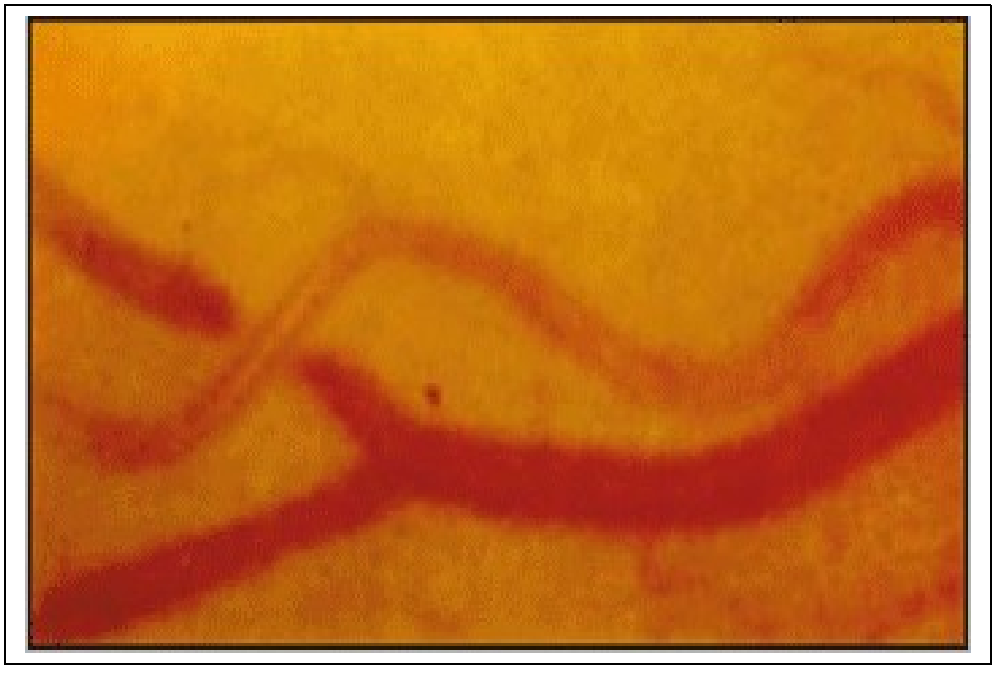
RETINAL VASCULAR CHANGES IN HYPERTENSION





GRADING OF RETINAL ARTERIOLOSCLEROSIS



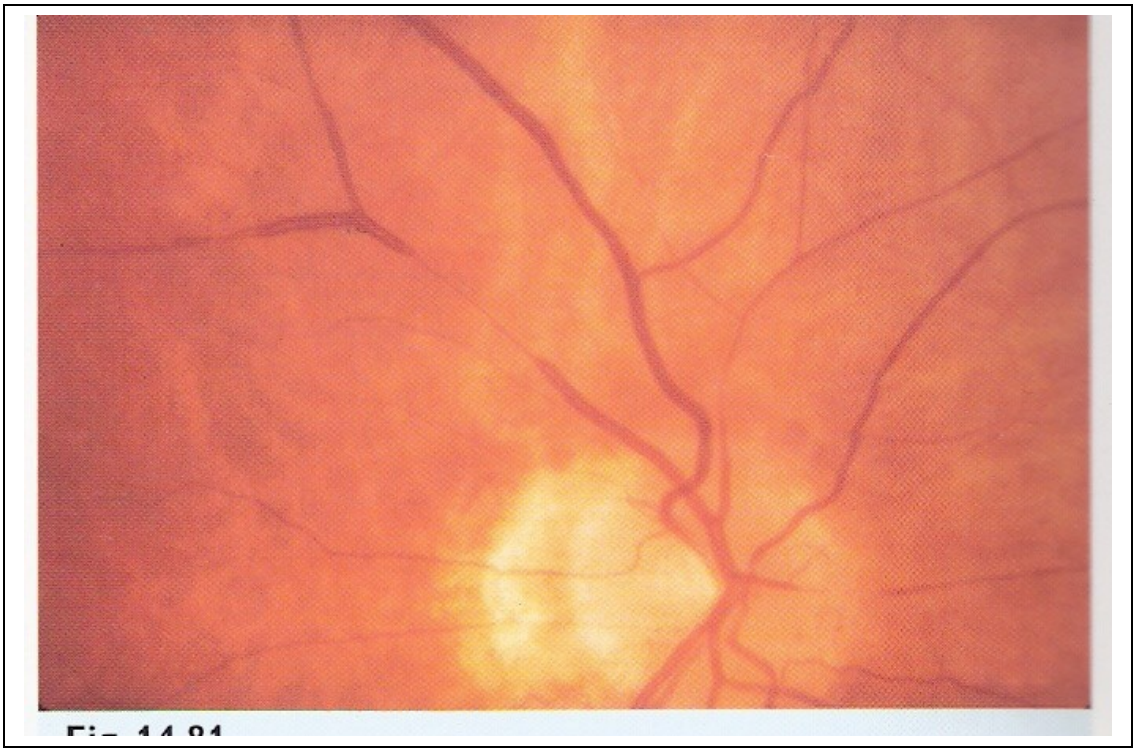


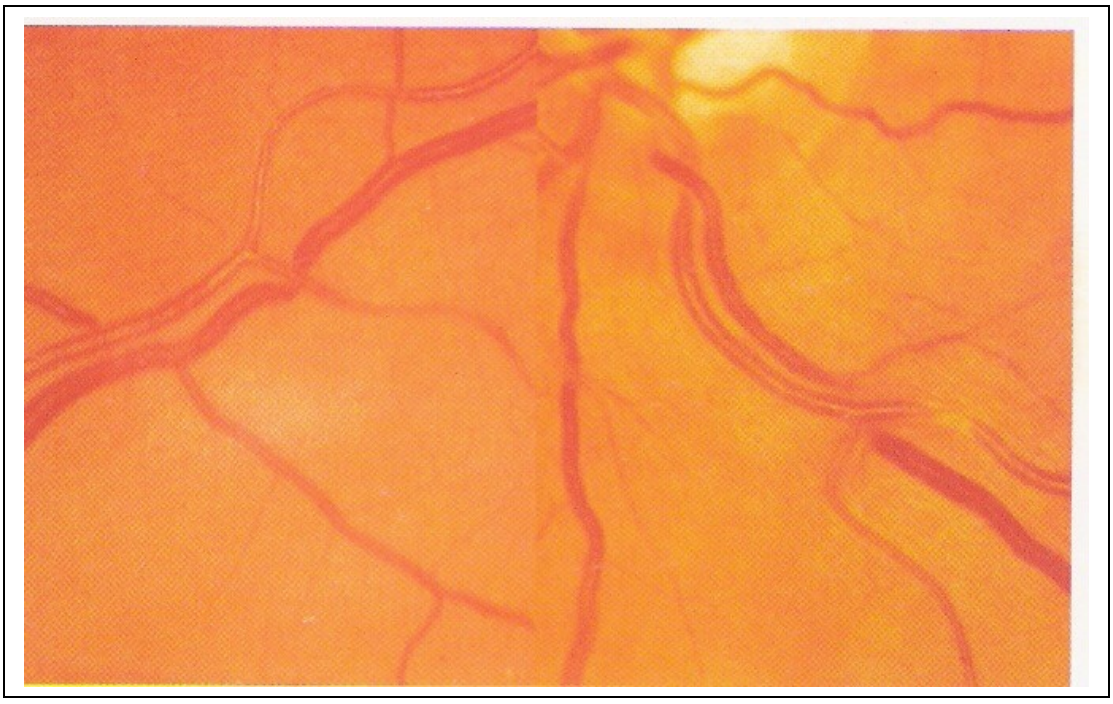
GRADE 1 HYPERTENSIVE RETINOPATHY

Generalised mild arteriolar narrowing

GRADE II HYPERTENSIVE RETINOPATY

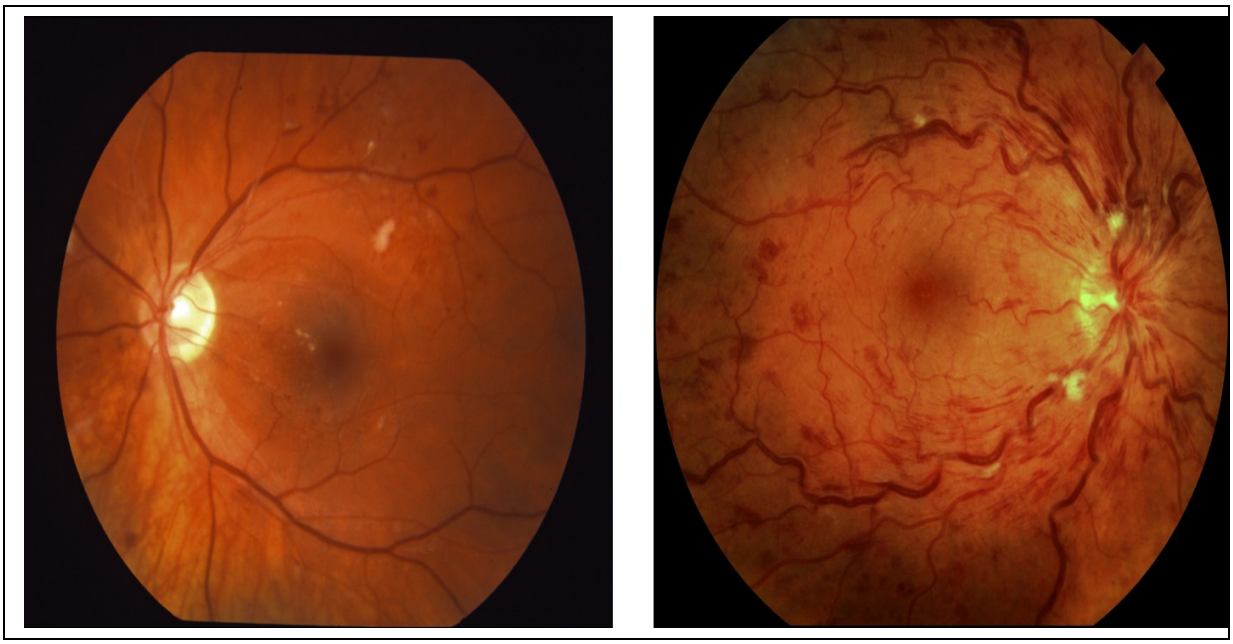
Marked arteriolar narrowing with focal constrictions





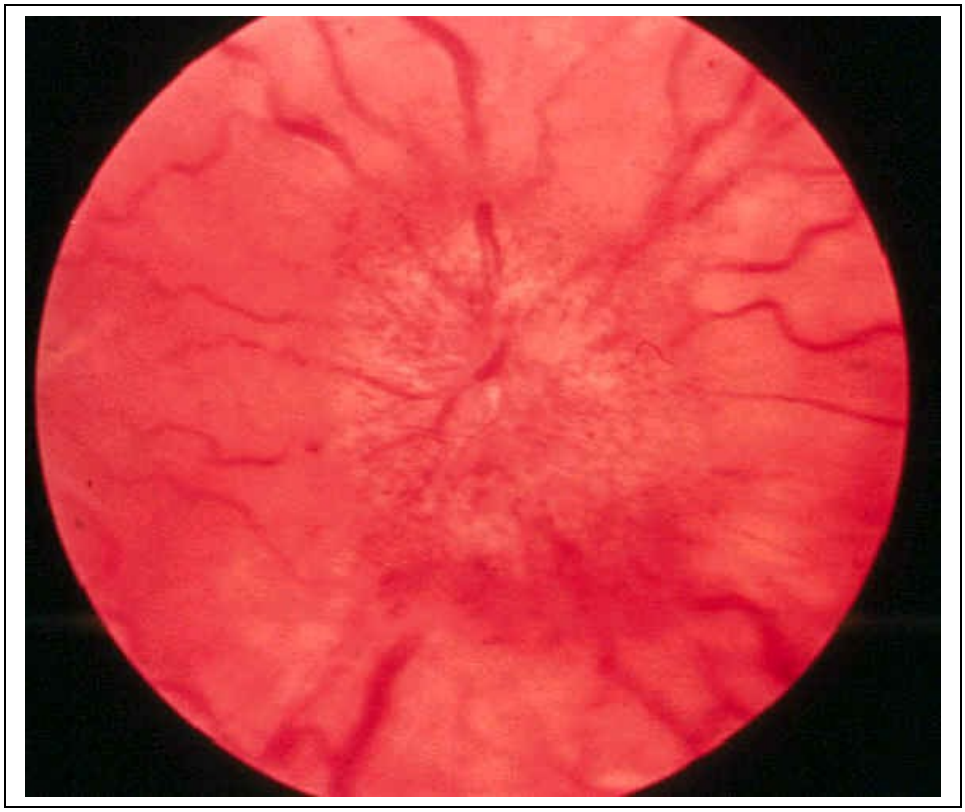
GRADE III HYPERTENSIVE RETINOPATHY

Stage II with haemorrhages, cotton wool spots and exudates)



GRADE IV HYPERTENSIVE RETINOPATHY

Disc Oedema



Macular star